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Clinical letter

Early post-operative convulsive status epilepticus in a patient with drug-refractory temporal lobe epilepsy and type I focal cortical dysplasia

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1. Introduction

Status epilepticus (SE) soon after epilepsy surgery is rarely described in case series.[1] When evaluating seizure outcome after surgery on the basis of Engel's classification, patients experiencing early SE may be overlooked. Underreporting early post-surgical SE does not allow the identification of SE early causative factors, and a definition of its prognostic value for long-term outcome.

We describe the case of a patient with drug-refractory temporal lobe epilepsy (TLE) who developed refractory SE immediately after an anterior temporal lobectomy (ATL).

2. Case report

In March 2009, a 31-year-old right-handed man was referred to our EMU for pre-surgical evaluation. His personal and family history were uneventful. No febrile seizures were reported.

Epilepsy onset was at age 20 years. Seizures were characterized by rising epigastric discomfort, *déjà-vu*, staring, oro-alimentary automatisms, right head deviation; no post-ictal deficits were reported. No other seizure types, either focal or generalized, were reported. A left cryptogenic TLE was diagnosed, and although various antiepileptic drugs (AEDs) were tried, the patient was never seizure free.

At hospital admission physical and neurological examinations were unremarkable. MRI was also normal. A cerebral fluorodeoxyglucose positron-emission tomography (FDG-PET) scan revealed an area of hypometabolism involving the lateral and mesial regions of the left temporal lobe, and long-term video-EEG monitoring revealed interictally focal slow and epileptic abnormalities located over the left temporal regions during sleep. Two ictal events were recorded during 50% AED withdrawal: one aura (rising epigastric sensation) did not show any clear ictal discharge, whereas a subsequent complex partial seizure clearly correlated with a left temporal ictal discharge (Fig. 1): left ATL of the dominant hemisphere was therefore indicated.

The surgical procedure was delayed by the patient until January 2012, when he underwent left ATL under propofol anesthesia. His AED treatment at the time was carbamazepine 600 mg bid and lacosamide 150 mg bid. The surgical procedure was concluded without any complications. Immediately after being awaken, the patient presented a generalized convulsive seizure that continued until he was again deeply sedated with midazolam, and early EEG revealed a continuous diffuse bilateral spike and wave discharge, predominating over the frontal regions (Fig. 1). The SE continued despite the subsequent use of loading doses of midazolam, diazepam, phenytoin and valproic acid. For this reason the patient underwent anesthesia with thiopental sodium. Neither a brain CT scan performed 6 h after surgery nor an MRI scan performed 24 h later showed any bleeding or ischemia.

The patient was kept under anesthesia for 14 days because of the reappearance of generalized tonic–clonic seizures whenever the treatment was lightened. The SE gradually subsided after loading doses of topiramate and phenobarbital. The patient was discharged from the ICU and transferred to our Neurology Unit 17 days after surgery.

At the time of final discharge one month after surgery, the patient's physical condition was normal and there were no neurological deficits; the EEG was characterized by rare slow waves in the left temporal regions. Neuropathological examination of the surgical specimens (Fig. 2) showed type IC focal

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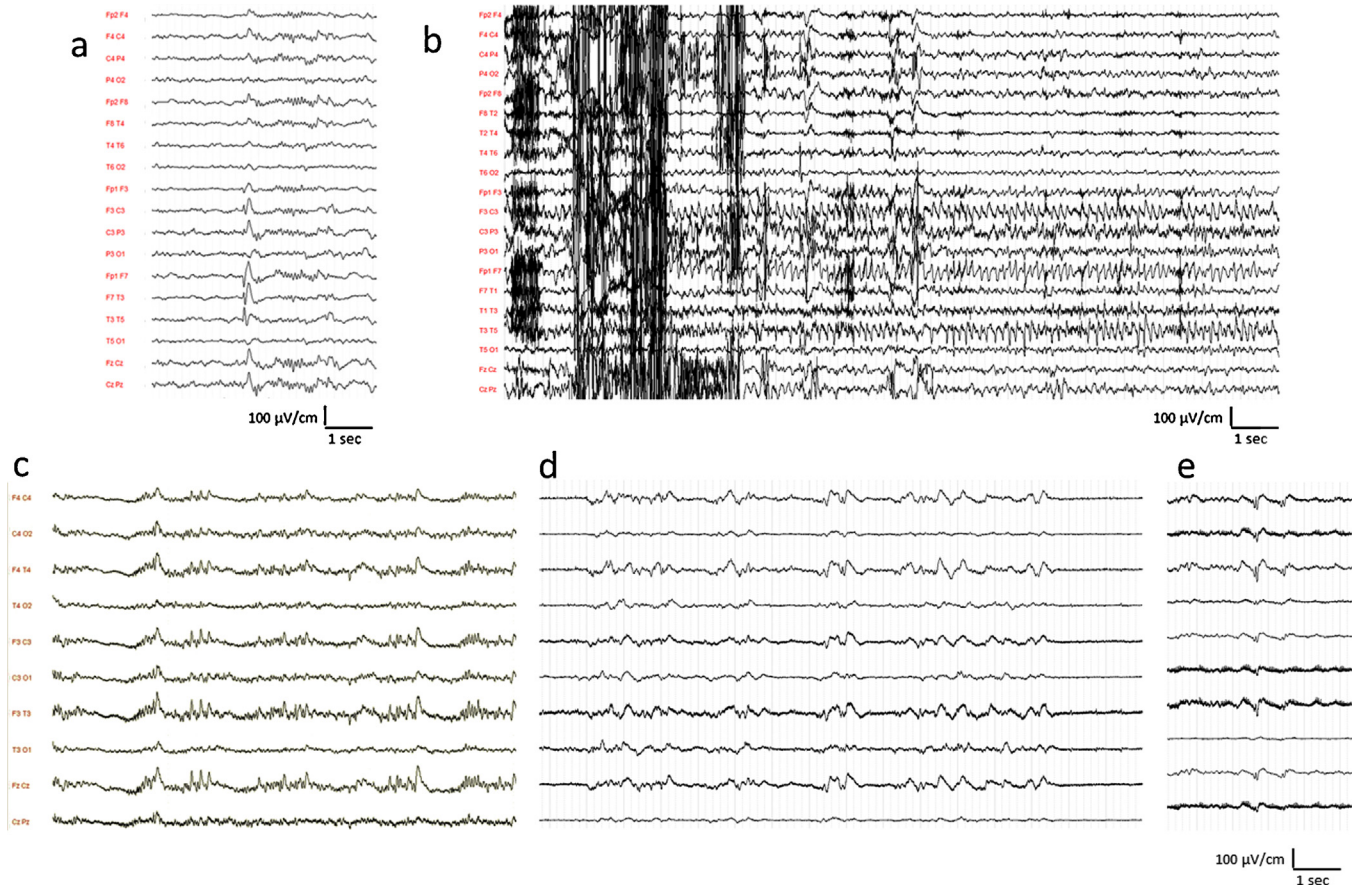

Fig. 1. Pre- and post-surgical EEG. Preoperative left temporal interictal high amplitude sharp epileptic transient (a) and ictal rhythmic discharge involving the left fronto-temporal regions (b). Early postoperative EEG (c) recorded a few minutes after the first convulsive seizure during midazolam administration showing diffuse pharmacological fast activities and bilateral spikes that are predominant over the anterior regions. (d) EEG three days after surgery during thiopental sodium (200 mg/h) administration showing diffuse slow waves, and spike and waves over the anterior regions, (e) slightly predominant on the right side.

cortical dysplasia (FCD). Now, after 19 months of follow-up, his post-surgical seizure outcome is classified as Engel's class Ib.

3. Discussion

SE has been rarely described as a possible outcome after ATL, but it is probably under-reported in surgical case series.

Published reports suggest that the possible causative factors of post-surgical SE include the co-existence of different epilepsies (focal and generalized),[2] multiple seizure types,[1] the withdrawal of AEDs, an extra-temporal location of the focus,[1] and, more convincingly, sub-total surgical removal of the dysplastic lesion, particularly when located in eloquent cortex as frequently happens in the case of type II FCDs.[3]

In our patient, the data obtained by non-invasive pre-surgical work up was consistent with the presence of an epileptic focus strictly localized within the left temporal lobe.

The patient was anesthetized using standard-dose propofol, and the entire surgical procedure was uneventful. All of the metabolic parameters were normal during and after surgery, and plasma AED levels were constantly monitored, and there were no significant variations. Brain scans performed after surgery, excluded ischemia, hemorrhagic lesions and other surgical complications.

The pathological examination of the surgical specimens did not reveal HS but type IC FCD declining in severity from the anterior to the posterior neocortex. The posterior limits of the resection showed a free margin clearly separating normal and dysplastic cortex (Fig. 2).

A recent study[1] has found *de novo* SE in 2.2% of 276 patients undergoing surgery because of drug-refractory focal epilepsy of temporal or extra-temporal origin.

In a previous paper,[3] the same group described three patients with SE starting immediately after the partial resection of a rolandic type IIB FCD: the authors hypothesized that the removal of the central balloon cell-rich part of the FCD may have freed the most epileptogenic periphery of the lesion from inhibitory control and led to SE and/or seizure worsening. This is probably not the case of our patient because of the apparently complete removal of the FCD.

A non-convulsive generalized absence SE in a patient undergoing ATL because of TLE with HS was attributed to a pre-existing generalized epilepsy, as suggested by the presence of interictal spike and wave discharges in the pre-operative EEG recording.[2] In our patient, the absence of any previous signs of generalized epilepsy rules out this possibility.

Other possible explanations, such as altered AED levels or surgical trauma, were also excluded.

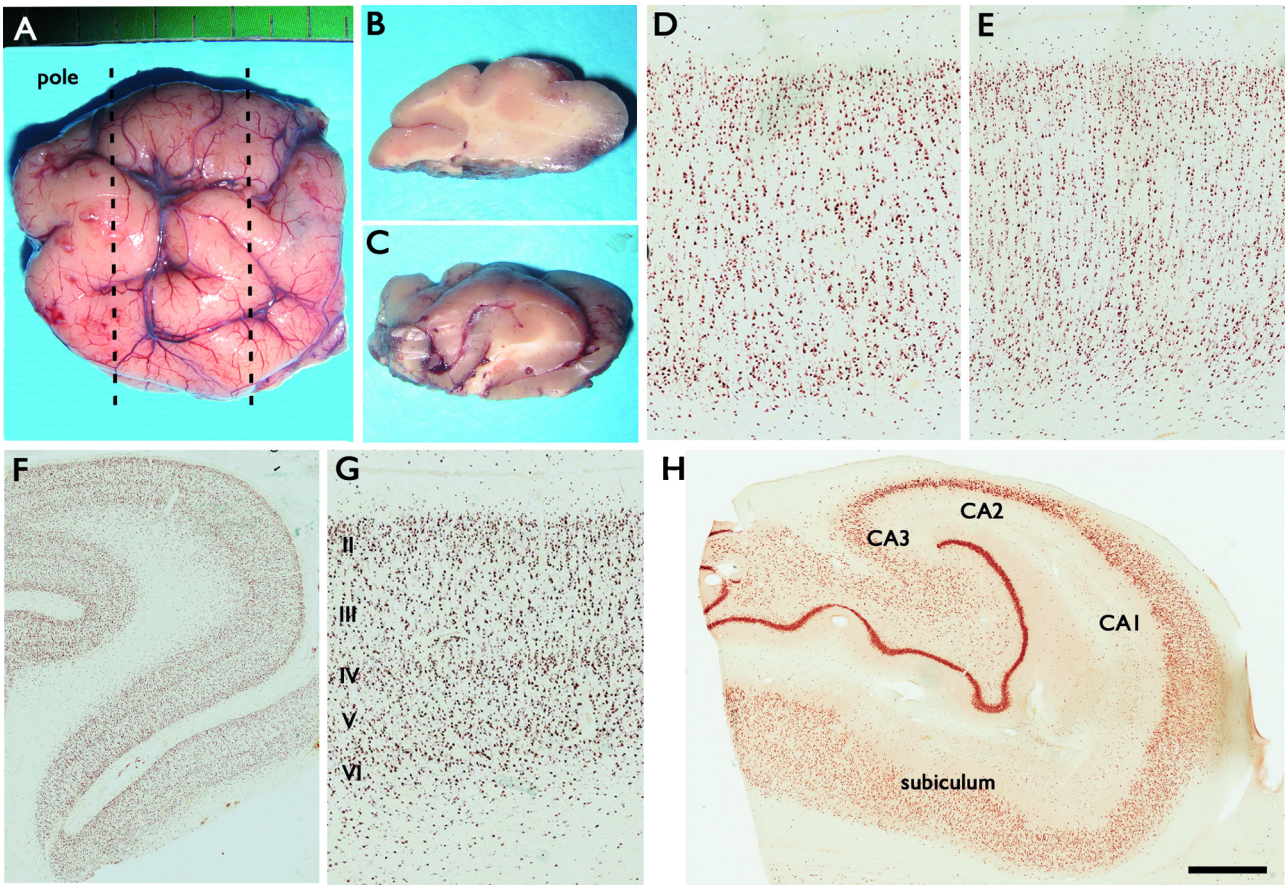

Fig. 2. (A) Left temporal pole. The specimen was oriented in accordance with the gyral patterns and cut from left (pole) to right in three slabs. There was no macroscopic evidence of structural abnormalities in the first (B) and third slabs (C). NeuN-immunostained sections obtained from the first tissue slab revealed architectural abnormalities characterized by abnormal horizontal (D) and vertical (E) cortical lamination, whereas the NeuN-stained section of the third slab at the edge of the resection showed normal cortex (F and G). No sclerosis was observed in the hippocampus (H). Bar: 1.3 cm (B and C); 400 μm (D and E); 2.3 mm (F); 630 μm (G), 1.3 mm (H).

4. Conclusion

We conclude that the appearance of generalized epileptic activity may have been driven by an imbalance in connectivity within an epileptic network that exceeded the anatomical limits of the lesion. The surgical removal of the dysplastic cortex may have activated epilepsy in predisposed, but still dormant, cortical areas, as recently suggested by modeling data: EEG epileptic discharges causing generalized or focal seizures may arise simply as a consequence of subtle changes in network structure and increased or decreased connectivity, with or without any localized pathological brain region,[4] possibly involving subcortical and brainstem structures.[5]

Conflict of interest

None of the authors has any conflict of interest to disclose.

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